available at www.sciencedirect.com
journal homepage: www.europeanurology.com

European Association of Urology

EAU Guidelines

EAU Guidelines for the Management of Urogenital Schistosomiasis

Karl-Horst Bichler, Ilya Savatovsky and the Members of the Urinary Tract Infection (UTI) Working Group of the Guidelines Office of the European Association of Urology (EAU): Kurt G. Naber (Chairman), Michael C. Bischop, Truls E. Bjerklund-Johansen, Henry Botto, Mete Cek, Magnus Grabe, Bernhard Lobel, Juan Palou Redorta, Peter Tenke*

Department of Urology, Hospital St. Elisabeth, St. Elisabeth Str. 23, D-94315 Straubing, Germany

Article info

Article history:
Accepted February 6, 2006
Published online ahead of print on February 28, 2006

Keywords:
Bilharziasis
Bladder cancer
Bulinus
Cervicitis
EAU guidelines
Endometritis
Epididymitis
Glycosaminoglycans
Metrifonate
Oxamniquine
Prziquantel
Salpingitis
Schistosoma haematobium
Schistosoma japonicum
Schistosoma mansoni
Schistosoma mekongi
Schistosomiasis
Urogenital tract infection

Abstract

More than 100 million people worldwide are affected by bilharziasis, caused by *Schistosoma haematobium*. For travellers precaution is most important. For the population in endemic areas, an integrated approach including health education is necessary. Effective pharmacologic treatment is available.

*Corresponding author. Tel. +49 9421 710 1700; Fax: +49 9421 710 1717.
E-mail address: kurt@nabers.de (K.G. Naber).

0302-2838/$ – see back matter doi:10.1016/j.eururo.2006.02.022

1. Introduction

More than 100 million people in the world, especially in rural and agricultural areas, are affected by bilharziasis of the urogenital tract, a parasitic disease caused by *Schistosoma haematobium* [1,2]. European urologists may see heavily infected migrants from endemic zones or tourists and development aid workers returning home with early stages of this disease.

2. Parasitic life cycle

S. haematobium is a parasitic trematode (flatworm). Humans are the main hosts for this parasite and both male and female worms reside in the pelvic venules. Fertilized eggs pass through the walls of the blood vessels into the tissues and the lumen of the rectum and sigmoid colon. They also pass into the urinary bladder, from which they are shed into the environment beginning a new life cycle. The fertilized eggs hatch in fresh water lakes, ponds, or rivers before developing into miracidia.

Miracidia migrate into a specific, intermediate host—the small snails of the genus *Bulinus*. The disease cannot spread beyond its natural habitat. The miracidia develop into sporocysts and after approximately 4 wk produce cercariae (by asexual multiplication), which infect the human host by penetrating unbroken skin during bathing, fishing, agricultural activities, or washing.

Once the skin is penetrated, the larvae migrate around the body via the circulatory system, reaching sexual maturity in about 6 wk. The adult worms may live for several decades in the venous plexuses around the pelvic organs, bladder, rectum, pelvic ureters, and deep genital organs. The worms lay eggs that migrate into these organs causing micro-mucosal perforation. Repeated micro-mucosal urothelial perforation causes haematuria. The eggs that are not secreted remain in the submucosa of the pelvic organs where they are encapsulated in fibrous granulomas.

The chronic lesions of schistomiasis depend on the extent of infection [3]. The disease is caused by the presence of the eggs rather than the worms themselves; the worms living in the veins are tolerated and do not lead to thrombosis.

3. Epidemiology

Schistosomiasis occurs in certain oases in southern Algeria, Morocco, and Tunisia, and in tropical Africa, between the latitudes of 35° North and 25° South, in the Savana and Sahel zones, while sparing the Sahara and the peri-equatorial forest belt. It occurs in Madagascar, Mauritius, all of the Nile valley, certain Middle Eastern countries, Yemen, Saudi Arabia, Iraq, and Iran. It has been eradicated in Lebanon and Israel. In the Far East, Australia, and South and Central America, bilharzial infections are mostly intestinal and caused by *S. mansoni*, *S. japonicum*, or *S. mekongi* according to the region [1,2,4].

The prevalence of the disease is closely linked to the educational and economic level of the population, the absence of adequate sanitation, and unprotected contact with contaminated fresh water. Bathing, swimming, and fishing are the main activities leading to infection. Frequency of contact is more important than duration of exposure.

4. Clinical features

Following cutaneous penetration by the cercariae, a localized itching may occur, or a rash may develop accompanied by fever. These early signs are quite often not noticed or entirely absent. Symptomatic disease begins months after the initial infection. Initial symptoms are dysuria, frequency, and haematuria. The migration of the parasite can cause mild fever, headache, dyspnea, and itchy rashes.

The chronic active phase of the disease is characterized by a massive increase in eggs in the urothelium, causing haematuria. Urinary tract obstruction (eg, due to vesical sclerosis or ureteral stenosis), renal insufficiency, and genital lesions are seen at a later stage of the infection. Typical symptoms in the later ulcerative stage are urethralgia, frequency, suprapubic pain, and haematuria [5,6].

Genital lesions such as epididymitis, salpingitis, endometritis, and cervicitis may cause sterility [7,8]. Vesical lesions will lead to inflammation, sclerosis, calcifications, loss of bladder capacity, bladder-neck stenosis, and later bladder cancer. Ureteral fibrosis and stenosis may lead to progressive deterioration in renal function, which may, ultimately, be totally lost [6].

Nephrotic syndrome develops sometimes in patients with *S. haematobium* infection. Severe renal changes are seen in up to 25% of patients with bilharzial infections [9]. Clinical symptoms are proteinuria and oedema. Renal biopsy specimens demonstrate mostly proliferative glomerular lesions.

Ectopic locations may be seen due to aberrant worm migration into the appendix, the spleen, and the spinal cord. In endemic areas, due to permanent reinfection, it is common to see lesions of different stages in the same patient [10]. Persistent haema-